

EDITORIAL COMMENT

3D Imaging of Device Leads

"Taking the Lead With 3D"*

Allan L. Klein, MD, Christine L. Jellis, MD, PhD
Cleveland, Ohio

Pacemaker lead-associated tricuspid regurgitation (TR) has been anecdotally described for many years, but investigation has been limited to a small number of observational studies (1). Overall, it seems that worsening of TR is common after transvenous lead placement (2). Perhaps the lack of formal research in this area has stemmed from our previous inability to adequately confirm that device lead placement was

See page 337

a true determinant of TR severity via 2-dimensional (2D) echocardiographic short-axis imaging (3). Serial assessment of TR may also be difficult because of dependence on loading conditions. Minimal TR at the time of implantation may appear more significant when the subject is not fasting and intracardiac filling pressures have normalized. Limited options available for lead repositioning or removal may have also fostered complacency regarding this issue. However, given the detrimental effect of significant TR on function (4) and survival (5) in a variety of conditions (6,7), any opportunity to minimize iatrogenic TR should be considered.

Three-dimensional (3D) echocardiography provides better spatial orientation and en face imaging of the tricuspid valve. The study by Mediratta et al. (8) in this issue of *JACC* makes timely use of this technique to assess the mechanism of device lead-associated TR. Perhaps surprisingly, only one previous small study has evaluated this concept (9).

Although observational, this study illustrates the benefit of routine 3D image acquisition because the investigators were able to identify 135 consecutive patients with complete 2D and 3D transthoracic echocardiographic imaging of the tricuspid valve with a device lead in situ. Despite the retrospective nature of the study, the findings show clinical relevance and mirror the practicalities of serial imaging. Technique feasibility with adequate 3D acquisition as well as off-line analysis was relatively good, with only 10% of subjects excluded from the analysis because of poor image quality. This appears to be better than would be typically expected in a busy clinical echocardiography laboratory with off-line processing capabilities. An advantage of this 3D study over the previous study is that there was a subgroup of subjects ($n = 53$) with adequate 2D echocardiograms before implantation, which facilitated direct comparison for worsening of TR after implantation.

Despite this being a study of 3D echocardiography application, this technique was reserved for structural assessment of valve leaflets and lead location only. Identification and severity grading of TR was performed via 2D color Doppler echocardiography. This likely reflects that 3D color Doppler imaging remains limited by low frame rates and resultant poor image quality. The authors chose to quantitate TR by using the largest vena contracta (VC) width after imaging in multiple views. They used mitral regurgitation VC classification, because VC grading for TR less than severe has not been well defined (10). A uniform Nyquist limit for VC measurement was not specified. Although there is consensus that this methodology is more accurate than jet area (11), it is still reliant on the jet being central and singular and may underestimate the severity of TR in the setting of eccentric or multiple jets. This may be problematic in this clinical setting,

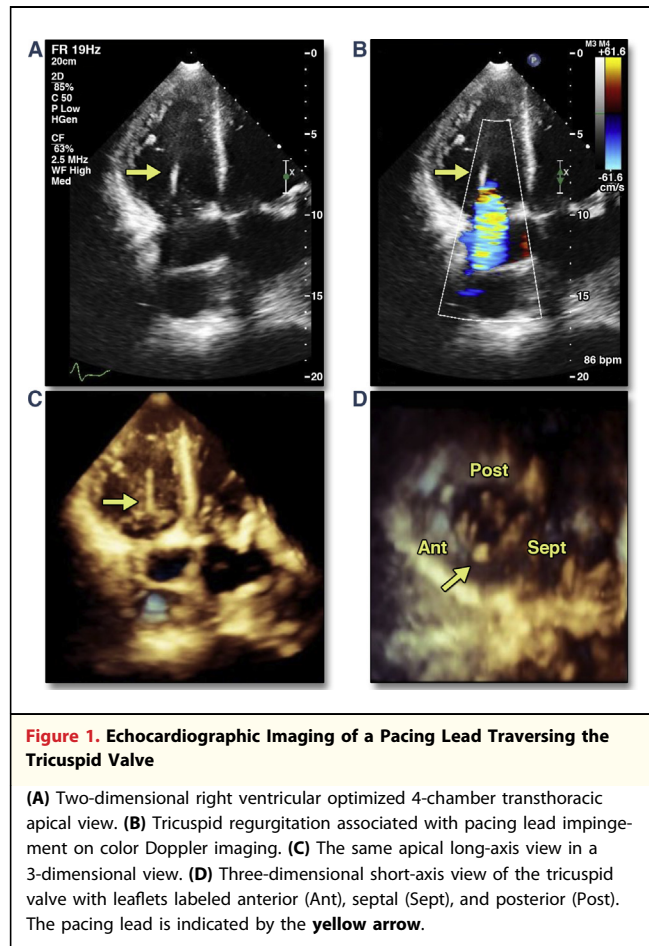
*Editorials published in *JACC: Cardiovascular Imaging* reflect the views of the authors and do not necessarily represent the views of *JACC: Cardiovascular Imaging* or the American College of Cardiology.

From the Heart and Vascular Institute, Cleveland Clinic, Cleveland, Ohio. Both authors have reported that they have no relationships relevant to the content of this paper to disclose.

where impingement of one valve leaflet may result in a highly eccentric regurgitant jet. Because of the absence of conclusive data regarding TR quantification, the current guidelines stipulate that multiple assessment parameters are preferred rather than a single modality (11).

The most significant results of this study include that 46% of subjects had device lead leaflet impingement prominently involving the posterior and anterior leaflets. This appears to be consistent with the typical position of the pacing lead tip adjacent to the septum, meaning that maximum curvature and lead displacement at the level of the valve is away from the septal leaflet. TR was predictably more associated with device leads causing valve impingement (VC: 0.6 ± 0.3 cm) rather than leads located centrally or in a commissural position (VC: 0.3 ± 0.3 cm) (Fig. 1). These findings were corroborated by the subgroup analysis, which showed that those with impinging leads had a greater increase in TR from baseline than those with nonimpinging leads, who had no appreciable change. Interestingly, lead type did not have an impact on the severity of TR; however, the majority (78%) had implantable cardioverter-defibrillator (ICD) or biventricular ICD leads, leaving only a small group with standard pacemaker leads for comparison. This contradicts previous work showing that ICD leads have higher rates of TR worsening than standard pacing leads (2), perhaps due to increased lead thickness and rigidity causing more valve impingement. This discrepancy may reflect underrepresentation of simple pacing leads in this study ($n = 27$; 22%). Biventricular quantification was omitted from this study but is clinically relevant. The high proportion of ICD implantations implies left ventricular dysfunction, so coexistent right ventricular dysfunction and dilation may have predisposed these subjects to TR due to reduced leaflet coaptation at baseline. Any alteration in valvular geometry may have been sufficient to potentiate TR in a susceptible cohort. Increased pulmonary pressures could likewise predispose certain subjects to the development of TR after implantation, whereas pre-existing TR may affect lead maneuverability.

The findings of this study suggest that the mechanism of TR inducement appears to be lead impingement or adherence rather than perforation ($n = 2$). This rate needs to be corroborated by autopsy or operative findings and may be center specific. 3D imaging resolution is not currently adequate to differentiate between lead impingement and adherence, while the propensity for lead-associated endocardial inflammation appears to be



unpredictable. Caution regarding lead extraction by traction or laser must be taken to avoid significant valvular disruption and further worsening of TR as well as issues related to sterility. Lead manipulation may be more feasibly associated with other concomitant cardiac surgery; however, resultant increased procedural times may augment the risk of infection. Some surgeons advocate securing the lead in a commissural position at the time of tricuspid valve annuloplasty and left-sided valve surgery. However, this is not a realistic proposition for patients with isolated lead-associated TR. Ultimately, there are currently no data to suggest that device lead extraction or repositioning has any impact on TR severity or outcomes in the total device population.

In this era of implantable device therapy, minimization of iatrogenic TR must be a central goal. Mediratta et al. (8) show useful application of 3D transthoracic echocardiography in the identification of leaflet impingement as the predominant cause of device lead-related TR. The long-term outcomes for this type of valvular dysfunction remain unknown, as

does the benefit of correction by lead extraction or repositioning. As 3D echocardiography techniques continue to evolve and improve, 3D color Doppler imaging at higher frame rates will likely provide complementary information regarding the real-time relationship of TR to device lead location. Intra-procedural 3D transesophageal or transthoracic echocardiographic guidance may prove useful for optimal lead placement and minimization of TR. However,

randomized verification against standard fluoroscopy is required, because protracted optimization of lead positioning could add to implantation time, risk of infection, and procedural costs.

Reprint requests and correspondence: Dr. Allan L. Klein, Cardiovascular Imaging J1-5, Heart and Vascular Institute, Cleveland Clinic, 9500 Euclid Avenue, Cleveland, Ohio 44195. *E-mail:* kleina@ccf.org.

REFERENCES

1. Al-Bawardy R, Krishnaswamy A, Bhargava M, et al. Tricuspid regurgitation in patients with pacemakers and implantable cardiac defibrillators: a comprehensive review. *Clin Cardiol* 2013;36:249–54.
2. Kim JB, Spevack DM, Tunick PA, et al. The effect of transvenous pacemaker and implantable cardioverter defibrillator lead placement on tricuspid valve function: an observational study. *J Am Soc Echocardiogr* 2008;21:284–7.
3. Lin G, Nishimura RA, Connolly HM, Dearani JA, Sundt TM III, Hayes DL. Severe symptomatic tricuspid valve regurgitation due to permanent pacemaker or implantable cardioverter-defibrillator leads. *J Am Coll Cardiol* 2005;45:1672–5.
4. Groves PH, Lewis NP, Ikram S, Maire R, Hall RJ. Reduced exercise capacity in patients with tricuspid regurgitation after successful mitral valve replacement for rheumatic mitral valve disease. *Br Heart J* 1991;66:295–301.
5. Nath J, Foster E, Heidenreich PA. Impact of tricuspid regurgitation on long-term survival. *J Am Coll Cardiol* 2004;43:405–9.
6. Gongora E, Dearani JA, Orszulak TA, Schaff HV, Li Z, Sundt TM III. Tricuspid regurgitation in patients undergoing pericardiectomy for constrictive pericarditis. *Ann Thorac Surg* 2008;85:163–70; discussion 70–1.
7. Kwak JJ, Kim YJ, Kim MK, et al. Development of tricuspid regurgitation late after left-sided valve surgery: a single-center experience with long-term echocardiographic examinations. *Am Heart J* 2008;155:732–7.
8. Mediratta A, Addetia K, Yamat M, et al. 3D echocardiographic location of implantable device leads and mechanism of associated tricuspid regurgitation. *J Am Coll Cardiol Img* 2014;7:337–47.
9. Seo Y, Ishizu T, Nakajima H, Sekiguchi Y, Watanabe S, Aonuma K. Clinical utility of 3-dimensional echocardiography in the evaluation of tricuspid regurgitation caused by pacemaker leads. *Circ J* 2008;72:1465–70.
10. Tribouilloy CM, Enriquez-Sarano M, Bailey KR, Tajik AJ, Seward JB. Quantification of tricuspid regurgitation by measuring the width of the vena contracta with Doppler color flow imaging: a clinical study. *J Am Coll Cardiol* 2000;36:472–8.
11. Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. *J Am Soc Echocardiogr* 2003;16:777–802.

Key Words: 3-dimensional echocardiography ■ intracardiac defibrillator ■ pacemaker tricuspid valve ■ tricuspid regurgitation.